

cessive sessions of ABPM on the same patients.

Conclusion: ABPM for 48 consecutive hours reveals a statistically significant pressor response that could reflect a novelty effect in the use of the monitoring device for the first time. This effect has marked implications in both research and clinical daily practice for a proper diagnosis of hypertension and evaluation of treatment efficacy by the use of ABPM.

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Circadian Variation of Blood Pressure in Normotensive and White Coat Hypertensive Subjects

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Background: The prevalence and clinical significance of white coat hypertension (WCH) is still controversial. Although recent longitudinal studies have provided preliminary prognostic data on subjects with WCH as compared to patients with sustained hypertension, the possible relation between WCH and vascular risk is still under debate. Accordingly, we compared the circadian pattern of blood pressure (BP) variability between normotensive subjects and patients with WCH.

Methods: We studied 332 subjects (129 mean), 43.9 ± 14.4 (mean \pm SD) years of age, with diurnal BP mean below 135/85 for systolic/diastolic BP. Among those subjects, 171 (74 men) had WCH (average of 6 conventional office BP measurements above 140 or 90 mm Hg for systolic or diastolic BP). BP was measured at 20-minute intervals during the day (07:00 to 23:00 hours) and at 30-minute intervals at night for 48 consecutive hours with a SpaceLabs 90203 ambulatory device. Circadian parameters established by population multiple-component analysis [Fernandez and Hermida. Chronobiol Int. 1998;15:191-204] were compared between normotensive and WCH subjects by non-parametric testing.

Results: Patients with WCH are characterized by a significant increase in systolic (2.5 mm Hg; $P < 0.001$) but not in diastolic BP ($P = 0.408$ for comparison of 24-hour mean) as compared to normotensive subjects. The differences in systolic BP between normotension and WCH are much more pronounced during the first 6 hours after awakening, and they are almost irrelevant during nocturnal resting hours. The largest and highly significant difference between groups was found around the clock in pulse pressure (about 3 mm Hg in 24-hour mean, $P < 0.001$).

Conclusion: In patients studied by 48-hour ambulatory monitoring, WCH is characterized by a significant elevation in systolic BP and, especially, in pulse pressure as compared to truly normotensive subjects. If indeed pulse pressure is an independent predictor of risk for cardiovascular events, WCH could then be associated to a long-term worst prognosis in comparison to true normotension, an issue that deserves further investigation.

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Blood Pressure Response During ABPM and Exercise and Cardiac Alterations

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Background: Some studies with a small sample size suggested that left ventricular mass index (LVMI) is more closely related to ambulatory blood pressure (ABPM) or exercise BP (ExBP) than to resting/casual BP.

Methods: In 1142 untreated patients (mean age 50.0 ± 9.2 y., 567 males) an ABPM (SpaceLabs 90207), a bicycle exercise test (50-100 watts, 5 min recovery period) and an echocardiography were performed within a 48 h period and thereafter the patients were divided into 4 groups (G_{1-4} ; table) due to their BP response.

Results: Despite a normotensive daytime BP during ABPM, patients of G_2 with an increased ExBP ($\text{ExBP}^+ > 200 > 100$ mmHg at 100 watts) revealed significantly ($p < 0.05$ - $p < 0.001$) higher values for LVMI, wall thickness (IVS, PWT) left ventricular enddiastolic dimension (LVID), left atrial size (LA) and a disturbed diastolic function ($\text{E/A} < 1$) compared to G_1 (normal ExBP, ExBP^-) and comparable values to G_3 (daytime ABPM hypertensive $> 135/85$, ExBP^+).

n=1142 x \pm 1s	ABPM normotensive, n=557		ABPM hypertensive, n=588	
	G_1 , ExBP- (n=241)	G_2 , ExBP+(n=316)	G_3 , ExBP- (n=47)	G_4 , ExBP+(n=538)
ABPM-daytime (mmHg)	121.0 \pm 7/ 76.7 \pm 5	124.9 \pm 7/ 79.9 \pm 4	141.4 \pm 6/ 93.1 \pm 5	147.7 \pm 10/ 95.8 \pm 7
ExBP, 100watts (mmHg)	177 \pm 24/90 \pm 11	220 \pm 20/ 114 \pm 11	193 \pm 21/ 105 \pm 11	228 \pm 21/ 120 \pm 11
LVMI (g/m 2)	70.3 \pm 18.6	85.7 \pm 19.0	84.2 \pm 17.3	90.9 \pm 22
LVID (mm)	49.1 \pm 4.9	50.5 \pm 5.9	49.6 \pm 5.2	50.1 \pm 5.7
IVS (mm)	8.3 \pm 1.5	10.0 \pm 1.6	9.7 \pm 1.7	10.4 \pm 2.1
PWT (mm)	7.1 \pm 1.5	8.6 \pm 1.1	8.5 \pm 1.4	8.9 \pm 1.4
LA size (mm)	32.8 \pm 5	37.8 \pm 6	36.4 \pm 5	38.0 \pm 6
E/A ratio	1.27 \pm 0.41	0.99 \pm 0.30	1.02 \pm 0.31	0.95 \pm 0.30

Conclusions: These data suggest that exercise BP may be an important determinant for cardiac alterations even in patients with a normotensive response during ABPM.

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Angiotensin Converting Enzyme Inhibitor Tissue Affinity Is Not Associated With Different Risk of Myocardial Infarction

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Background: Angiotensin converting enzyme (ACE) inhibitors may decrease the risk of myocardial infarction (MI), independent of blood pressure lowering, by attenuating local angiotensin-mediated atherogenic effects in vascular tissue. ACE inhibitors differ in their affinity for tissue-bound ACE and some have hypothesized that this could have differential effects on MI risk.

Methods: Data were obtained from two case-control studies of first MI, ages 30 through 75, conducted among 68 hospitals in an 8-county area during a 5 year period. Cases were hypertensives hospitalized with a first MI, and controls were hypertensives randomly selected from the same geographic area. Detailed information regarding medication use and other clinical and demographic data were obtained by telephone interview. ACE inhibitors were categorized into high (benazepril, quinapril, ramipril) and low (lisinopril, enalapril, fosinopril, captopril) tissue ACE affinity groups based on previously demonstrated tissue ACE potency.

Results: Among the 851 cases and 1791 controls who participated, there were 174 high and 566 low affinity tissue ACE inhibitor users. After adjustment, using multivariable logistic regression, for age, sex, race, study source, physical activity, quantity smoked/day, body mass index, family history of MI, other antihypertensive use, and history of diabetes, hypertension, or hypercholesterolemia, the odds ratio (OR) for MI among current high affinity ACE inhibitor users compared with low affinity users was 0.85 (95% CI: 0.58, 1.24; $P = 0.39$). In addition, increasing tissue ACE potency was not associated with an altered risk of MI (P for trend = 0.40).

Conclusion: ACE inhibitor tissue affinity did not appear to affect MI risk in this study. Therefore, MI protection seen in clinical trials of specific ACE inhibitors may be generalizable to all agents in this class. Further research is needed to evaluate the effects of dose on drugs with different tissue-ACE affinities and to assess other potential differences among ACE inhibitors.

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High Prevalence of Functioning Adrenocortical Lesions in Low Renin Arterial Hypertension

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Background: Recent reports suggest a higher than expected prevalence of states of increased adrenocortical function in arterial hypertension. Parallely, non functioning adrenal lesions have been increasingly detected due to a growing number of imaging studies, leading to frequent problems of differential diagnosis. Aim of the study was to investigate the presence of laboratory features of mineralocorticoid excess and their possible association with anatomical abnormalities of the adrenal glands in hypertensive patients with a previous diagnosis of "low renin" essential hypertension.

Methods: 20 low renin hypertensive patients were selected for adrenal CT according to the presence of borderline/high aldosterone levels together with reduced/suppressed PRA. Adrenocortical scintigraphy with ^{131}I -iodocholesterol and ACTH suppression by oral desametasone was performed in 11 patients with positive CT findings. Preliminary results on saline loading test (2 liters/4 hours) were also obtained in 9 patients.

Results: CT was positive in 11 subjects, showing either bilateral (n=3) or unilateral (n=8) adrenal lesions. Iodocholesterol scintigraphy identified functioning lesions in 7/11 patients. In the 9 patients who underwent the test saline loading increased both systolic ($p < 0.02$) and diastolic ($p < 0.01$) blood pressure levels, decreased aldosterone ($p < 0.01$) and increased ANP levels ($p < 0.01$). Aldosterone and ANP levels significantly related to each other ($p < 0.05$) and to blood pressure levels ($p < 0.02$, $p < 0.01$ respectively).

Conclusions: The results, to be confirmed in larger series of cases, suggest a high prevalence of functional adrenal lesions in low renin hypertensive patients with laboratory features of even mild/moderate mineralocorticoid excess. In this context, adrenocortical CT and scintigraphy are particularly useful, allowing a definite diagnosis and correct therapeutic approach. The preliminary results of the saline test indicate features of salt sensitivity of blood pressure in these hypertensive forms.

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Electrocardiographic Strain Pattern and the Prediction of Cardiovascular Morbidity and Mortality in Hypertensive Patients: The LIFE Study

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Background: The ECG strain pattern of ST depression and T-wave inversion is a marker for left ventricular hypertrophy (LVH) and adverse prognosis in population studies. However, whether ECG strain is an independent predictor of cardiovascular (CV) morbidity and mortality in the setting of aggressive antihypertensive therapy is unclear.

Methods: ECGs were examined at study baseline in 8,854 hypertensive patients with ECG LVH by Cornell voltage-duration product and/or Sokolow-Lyon voltage on a screening ECG who were treated in a blinded manner with atenolol- or losartan-based regimens and followed for at least 4 years (mean 4.8 ± 0.9) in the Losartan Intervention For Endpoint reduction in hypertension (LIFE) study. Strain was defined as a downsloping convex ST segment with inverted asymmetrical T-wave opposite to the QRS axis in leads V5 or V6.

Results: In pre-specified analyses, the ECG strain pattern was present in 971 patients (11.0%) and the LIFE composite endpoint of CV death, non-fatal myocardial infarction (MI) or stroke occurred in 1,035 patients. In univariate Cox analyses, ECG strain was a